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A Neural Network Approach to Obsessive–Compulsive Disorder

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A central methodological innovation in cognitive science has been the development of connectionist or neural network models of psychological phenomena. These models may also comprise a theoretically integrative and methodologically rigorous approach to psychiatric phenomena. In this paper we employ connectionist theory to conceptualize obsessive–compulsive disorder (OCD). We discuss salient phenomenological and neurobiological findings of the illness, and then reformulate these using neural network models. Several features and mechanisms of OCD may be explicated in terms of disordered networks. Neural network modeling appears to constitute a novel and potentially fertile approach to psychiatric disorders such as OCD.

In recent decades the psychological sciences have witnessed a "cognitive revolution." Behaviorism, which was the central paradigm of American psychology in the first part of the century, has given way to the multidisciplinary field of cognitive science. The component disciplines of this field include cognitive psychology, artificial intelligence, linguistics, anthropology, and philosophy (Gardner, 1985; Posner, 1990; Stillings et al., 1987). The term "cognitive" itself denotes a somewhat restricted set of phenomena, and cognitive science has indeed been criticized for excluding the processing of affect (Oatley, 1987), and for insufficient attention to the context of information-processing (Norman, 1993). Nevertheless, the broad spectrum of concepts and methodologies embraced by cognitive science is attractive to those interested in comprehensive and integrative approaches to the mind, and recent cognitive science has attempted to address the lacunae of early sim-

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plistic information–processing approaches (Lakoff, 1987; Varela, Thompson, and Rosch, 1991).

Central to cognitive science is the use of computational models of the mind. In these models, cognitive architecture is typically specified in either symbolic or connectionist terms (Posner, 1990). The elements of symbolic systems are symbols, which are stored in associative structures. The elements of connectionist systems are simplified and schematized neurons which are interconnected in a neural network. While there are those who argue that only one or the other of these two approaches is useful, it is also possible to conceive of them as complementary — with schema architectures allowing a top–down approach, and connectionist architectures being more suited to a bottom–up approach to the mind. Neural networks can be conceptualized as providing a detailed, neurobiologically consistent formalization of many of the properties of schemas (Rumelhart, Smolensky, McClelland, and Hinton, 1986).

In recent years connectionist models have been increasingly used to model a variety of psychological phenomena (Anderson and Rosenfeld, 1988; Reeke and Sporns, 1993; Rumelhart and McClelland, 1986). Neural network models appear to offer several positive characteristics. In particular, the conceptual apparatus of connectionism seems to integrate research in computational science and neuroscience. Thus, the parallel distributed processing of computational networks is characterized by several features that are also seen in biological networks. These include information-processing in the absence of central control; information-processing in parallel rather than in series; content addressable memory rather than memory located by an abstract name; relatively slow processing in individual units in contrast with rapid processing in whole networks; processing of information by different areas of interacting modules using reciprocal connections; learning via adjustment of synaptic strengths; efficient pattern recognition; and graceful degeneration rather than sudden crashing of systems after disturbances to the network (Norman, 1986). In addition, computational implementation of neural networks seems to offer a rigorous methodology for cognitive science.

One of the strongest arguments in favor of the integrative potential of cognitive science, and its incorporation of such complexities as affect and context, is the increasing application of cognitive science approaches to understanding disorders of the mind (Stein, 1993; Stein and Young, 1992). While much of this work has employed symbolic approaches, several neural network models of clinical disorders have also been developed. The work of Hoffman (1987) and Cohen and Servan–Schreiber (1992) on connectionist models of schizophrenia has been pioneering, and may be argued to constitute a novel methodology for psychiatry. There has also been preliminary connectionist work on bipolar disorder (Hestenes, 1992), anxiety, and depression (Williams and Oaksford, 1992).

In this paper we build on early work on neural networks of clinical disorders, and attempt to construct a connectionist model of obsessive–compulsive disorder (OCD). In the past, OCD has been understood primarily in psychoanalytic and behaviorist terms, while more recently a biological perspective has predominated (Stein and Hollander, 1992). In this paper we suggest that a cognitive science approach that draws on neural network theory is consistent with the phenomenological and neuropsychiatric features of OCD, and with our knowledge of its neurobiological mechanisms. We begin by outlining the central features and mechanisms of OCD.

Neurobiology of OCD

Recent neurobiological research on OCD has focused on both the neurochemistry and the neuroanatomy of the disorder (Hollander, DeCaria, and Liebowitz, 1989). Neurotransmitter research was encouraged by the finding that OCD, unlike disorders such as depression and panic, has a better response to serotonin reuptake blockers than to noradrenaline reuptake blockers (Zohar and Insel, 1987). Early work demonstrated that in at least a subgroup of OCD patients there are increased cerebrospinal fluid levels of the serotonin metabolite 5-hydroxyindoleacetic acid (CSF 5-HIAA), and that levels fall during effective pharmacotherapy (Thoren et al., 1980).

More recently, studies employing pharmacological challenge paradigms have further supported the hypothesis that serotonin mediates OCD. Thus methyl-chlorophenylpiperazine (mCPP), a serotonin agonist, leads to increased OCD symptoms in a subgroup of patients, and is associated with blunting of neuroendocrine response in patients compared with controls (Hollander, DeCaria, Nitescu et al., 1992; Zohar, Mueller, Insel, Zohar–Kadouch, and Murphy, 1987). Furthermore, after treatment with serotonin reuptake blockers, administration of mCPP is no longer followed by behavioral exacerbation or neuroendocrine blunting (Hollander, DeCaria, Gulley et al., 1991; Zohar, Insel, Zohar–Kadouch, Hill, and Murphy, 1988).

Other neurotransmitters may, however, also be involved in OCD. Patients who are refractory to serotonergic medications may, for example, respond to dopamine blocker augmentation, particularly when schizotypal personality traits are present (McDougle et al., 1990). Chronic administration of dopamine agonists may lead to stereotypes in animal models, and may lead to exacerbation of OCD in humans (Goodman, McDougle, Price, Riddle, Pauls, and Leckman, 1990). Noradrenergic and other systems may also play a role in OCD, but the evidence for this is more equivocal (Hollander, DeCaria, Nitescu et al., 1991).

The possibility that OCD has a specific underlying neuroanatomy was raised by early reports that OCD can be a sequela of neurological insults.

More recent controlled studies have confirmed the comorbidity of OCD and certain neurological disorders (Stein, Hollander, and Cohen, in press; Swedo et al., 1989), and have demonstrated increased neurological soft signs in a subgroup of OCD patients (Hollander, Schiffman et al., 1990). Treatment studies once again provide specific clues for neurosurgical interruption of frontal lobe — basal ganglia pathways lead to improvement in OCD symptoms (Martuza, Chiocca, Jenike, Giriunas, and Ballantine, 1990). Indeed, it appears that neurological insults to the frontal lobe and basal ganglia in particular may be associated with OCD (Khanna, 1988; Rapoport and Wise, 1988; Stein, Hollander, and Cohen, in press).

Imaging studies have provided further evidence that frontal lobe and basal ganglia play a role in OCD. Structural studies with computed tomography (CT) have found decreased caudate volume in OCD (Luxenberg, Swedo, Flament, Friedland, Rapoport, and Rapoport, 1988), although a subsequent study did not replicate this finding (Stein, Hollander, Chan, DeCaria, Hilal, Liebowitz, 1993). Functional studies with single photon emission tomography (SPECT) and positron emission tomography (PET) have found that patients with OCD have increased metabolism in frontal cortex (Baxter, Schwartz, Guze, Bergman, and Szuba, 1990).

Psychology of OCD

Despite progress in neurobiological knowledge of OCD, there remains the question of how neurochemical and neuroanatomical perturbations lead to the specific psychological characteristics of OCD. These characteristics cannot, of course, simply be reduced to physical perturbations. A full understanding of OCD requires attention to the emergent psychological characteristics of the disorder. In particular, we need to consider the central phenomenological features of OCD and their corresponding functional deficits.

Current diagnostic classifications of OCD (American Psychiatric Association, 1987; World Health Organization, 1992) emphasize that OCD is characterized by the presence of obsessions and/or compulsions. On this view, obsessions are defined as intrusive thoughts, images, or impulses that are experienced, at least initially, as senseless or inappropriate. Compulsions, on the other hand, are repetitive actions or mental rituals that are performed in response to an obsession, or in a stereotyped way.

Although similar descriptions of obsessions and compulsions have been provided by authors of various theoretical persuasions (Freud, 1909; Jaspers, 1963; Marks, 1987), the phenomenology of OCD can be surprisingly heterogeneous (Rasmussen and Eisen, 1989). It is notable that OCD was initially classified as a form of insanity, and only later as a neurotic disorder (Berrios,

1985). The former classification suggests a lack of insight and resistance, the later classification emphasizes the presence of these features in OCD. Nevertheless, such features as insight and resistance vary from patient to patient (Rasmussen and Eisen, 1989).

Different emphases in the phenomenological characterization of OCD doubtless reflect divergent theoretical assumptions and perspectives. Freud, for example, emphasized the spectrum between character disorder and obsessional neurosis, in line with his view of the latter as a distortion of normal personality development. Similarly, he emphasized conflict as an important feature of obsessional symptomatology, so reflecting his hypothesis that psychic conflict is instrumental in the etiology of obsessional neurosis. Modern classifications draw a firm distinction between OCD and obsessive compulsive personality disorder, perhaps in line with an implicit biologically influenced view that OCD is caused by a specific and discrete lesion. In line with behavioral theory, compulsions are seen as performed in response to obsessions.

Our aim here is to go beyond reductionistic biological or behavioral approaches to OCD. We therefore need to emphasize other aspects of the phenomenology of the disorder. Rasmussen and Eisen (1993) have used factor analysis to delineate a number of core features of OCD in addition to obsessions and compulsions. One core feature of OCD can be characterized in terms of a sense of incompleteness. OCD patients may, for example, describe the feeling that they must continue a ritual until things feel "just so" or "just right." Until that point, their actions are subjectively experienced as incomplete. Lopez–Ibor (1990) has also noted that feelings of incompleteness are an important phenomenological component of OCD, and has suggested that descriptions of this component can be traced back to the work of Janet (1911) and others.

A second core feature of OCD detailed by Rasmussen and Eisen (1993) is abnormal risk assessment or harm avoidance. OCD patients may be preoccupied with the thought that something terrible will happen. This may be accompanied by a sense of doubt and uncertainty. Lopez–Ibor (1990) also describes doubt as a core component of OCD, and once again traces early descriptions of this in the psychiatric literature. The term *maladie du doute* was a common name for obsessional states in the last century (Berrios, 1985). Although this feature of OCD has received some empirical attention, its subjective nature has perhaps contributed to a relative lack of research interest.

These two components of OCD symptomatology can be employed as key facets in a theoretical position on the nature of the functional deficits in OCD. In line with previous cybernetic approaches (Pitman, 1987; Rapoport and Wise, 1988) to OCD, we have suggested that OCD involves impairment in the determination or assessment of goal–response completion (Stein and Hollander, 1992). Thus, in some patients a deficit in match–mismatch mech-

anisms may result in inadequate determination of goal discrepancy with repetitive behaviors prior to goal completion (Liebowitz and Hollander, 1991). In other patients, inadequate assessment of goal discrepancy with overestimation of the harm associated with possible mismatch may result in exaggerated uncertainty and doubt.

The validity of these putative deficits is provided some support by neuropsychological research. We have found that the Matching Familiar Figures Test (MFFT), for example, is useful in detailing the heterogeneity of OCD (Hollander, Liebowitz, and Rosen, 1991). This test of reflection–impulsivity involves comparing a set of detailed figures with a background foil which differs in only one detail. One subgroup of OCD patients responds rapidly with a high error rate. A second subgroup of OCD patient responds slowly and with a low error rate. It may be postulated that the first group has difficulty in determining goal–response completion, while the second group is characterized by harm overestimation.

Furthermore, there is preliminary evidence that neuropsychological responses on the MFFT correlate with specific neurobiological deficits (Hollander, Liebowitz, and Rosen, 1991). Thus rapid erroneous response correlates with increased neurological soft signs and with poor response to serotonin reuptake blockers. On the other hand, slow correct response correlates with more reactive responses to serotonergic challenge. Thus, there is some suggestion that one subgroup of OCD patients has match–mismatch impairment that is associated with neurological deficits, while a second subgroup of OCD patients has harm overestimation that is associated with serotonin dysregulation (Stein and Hollander, 1992).

This interpretation of the data should, however, be considered preliminary. Particular information processing dysfunctions may represent strategies for dealing with quite varied underlying neurobiological impairments. Furthermore, different neurobiological impairments in OCD may represent dimensional deficits rather than discrete subgroups. Nevertheless, this preliminary interpretation does provide a useful and integrative heuristic for further research on OCD.

Neural Networks

Neural networks can be described in terms of their processing units, the way these are put together, and the way in which they learn (Hanson and Burr, 1990; Williams and Oaksford, 1992). We briefly discuss each of these topics in turn.

The units of neural networks comprise schematized neurons. Like neurons, each unit has inputs (dendrites) from other units, and outputs (axons) to other units. Each input has a particular weight (synapse), which can be posi-

tive (excitatory) or negative (inhibitory). The sum of these weights is the net input. Whether or not the unit is activated (potentiated) depends upon the net input and the unit's activation function. Activation functions differ in different units. In the simplest case there is a binary threshold; if the unit is activated it turns on (1), if not it stays off (0) [as in the case of the McCullough–Pitts neuron]. A more complex case is the sigmoid function, which produces a continuous output between 0 and 1.

The topology of a unit is the way in which units are joined to one another. In a totally connected network, such as the Hopfield network (Hopfield, 1982, 1984), all units are connected to one another. In a feedforward unit, information flows in only one direction, from input units to output units. In multilayer networks, there are also hidden units between input and output units. Whereas early pioneers of connectionism focused more on simple networks, computer implementation of neural networks has allowed a shift to more complex ones.

Learning takes place in networks via modification of synaptic weights. Neural networks, for example, can be trained to associate particular input patterns with particular output patterns. During training, input patterns are presented and synaptic weights are changed according to a learning rule. In a multilayer network, error can be measured across the output units and then compensatory changes can be made at each level of the network (back-propagation).

How are memories stored in a network? Many networks can be conceptualized as constraint networks in which each unit represents an hypothesis (i.e., a feature of the input), and in which each connection represents constraints among the hypotheses (Rumelhart et al., 1986). A variation of Hebb's rule, for example, states that if feature A and B often co-exist, then the connection between the two will be positive. On the other hand, when the two features exclude one another, then the connection will be negative. Inputs may also be conceptualized as constraints. When the network runs, it settles into a locally optimal state in which as many as possible of the constraints are satisfied. Hopfield showed that if Hebb's rule is followed in his fully connected network then local computational operations, in which unit adjusts its activation up or down on the basis of its net input, allow the network to converge toward states that maximize a global measure of constraint satisfaction.

The information processing of a network from state to state can be conceptualized in terms of movement over a goodness-of-fit landscape (Rumelhart et al., 1986). The system processes input by shifting from state to state until it reaches a state of maximal constraint satisfaction, that is, it climbs upward until it reaches a goodness maxima. A landscape can be described in terms of the set of maxima which the system can find, the size of the region that feeds

into each maximum, and the height of the maximum itself. The positions of the system correspond to the possible interpretations, the peaks in the space correspond to the best interpretations, the extent of the foothills surrounding a particular peak determines the likelihood of finding the peak, and the height of the peak corresponds to the degree that the constraints of the network are actually met (Rumelhart et al., 1986).

The particular maximum that the system reaches is determined by where the system starts and by the modifications of the system induced by the input. Stored positions can be seen as acting as "attractors" that pull the system into their activation patterns. If external inputs cause the network to move into a position that is similar to a stored position, the system will then climb to that interpretation in its entirety.

Clearly, it is possible for a network to settle on a local maximum that is not in fact the highest maximum (best interpretation). Some networks therefore include random perturbation of states. This allows the network to move down a maximum, increasing the chance that the highest maximum will be climbed. Ackley, Hinton, and Sejnowski (1985), for example, described a network with stochastic adjustments to individual units (the Boltzmann network). This adds to the network a process of simulated annealing, which allows the network to search for the global maximum. Similar additions and modifications have led to the construction of increasingly complex and powerful network models in the past few years.

While it is possible to provide sophisticated mathematical formalizations of a goodness-of-fit landscape, this way of characterizing neural networks was emphasized here because of its intuitive appeal. For example, cognitive scientists often employ the notion of schemas, prototypical abstractions that develop from past experience and that guide the organization of new information (Thorndyke and Hayes–Roth, 1979; Stein, 1992b). Schemas allow rapid processing of information, but also result in typical biases (Winfrey and Goldfried, 1986). The notion of a goodness-of-fit landscape can readily be used to reframe schema theory. Thus a particular neural network, prompted by a given set of data, rapidly moves toward a previously acquired landscape. This allows rapid information processing, but again, may result in certain distortions (Rumelhart et al., 1986).

Neural Networks in OCD

To describe neural networks that can model the neurobiological and psychological features of OCD, we draw on networks that have already been discussed in the connectionist literature. We first discuss a network that relates to the inadequate determination of goal discrepancy, and we then consider a network for the assessment of goal discrepancy as harmful.

A network for determining goal discrepancy might begin with work on the frontal lobes, which have been described as the "executive of the brain" (Pribram, 1973). While several neural network models of frontal lobe damage have been constructed (Levine, Leven, and Prueitt, 1992), the one that perhaps best combines psychopathology, neuropsychology, and neurobiology is that of Cohen and Servan–Schreiber (1992). Their model was constructed in order to depict the processing of context and its disturbances in patients with schizophrenia. It addresses (a) the phenomenology of context processing, (b) the neuropsychology of context processing, and (c) the biology of context processing.

Cohen and Servan–Schreiber define context as information that is relevant to, but not part of the content of a behavioral response. This can be task instructions or specific previous stimuli that determine correct behavior. Neuropsychological tests that measure the processing of context include the Stroop task and the Continuous Performance Test (CPT). Lastly, Cohen and Servan–Schreiber suggest that dopamine tracts in prefrontal cortex are responsible for the representation of context. Central to their networks is a module for the representation of context. The units of the module are governed by a gain parameter (simulating the effects of dopaminergic activity). These networks model the behavior of schizophrenic patients on context processing tasks.

We would argue that each of these features of Cohen and Servan–Schreiber's model also applies to the determination of goal discrepancy. First, both the processing of context and the determination of goal discrepancy require a focus on information relevant to but separate from a behavioral task. The representation and maintenance of context can be seen as a component of goal-feedback tasks. Second, tests such as the Stroop task, the CPT, and the MFFT all measure the ability of subjects to determine goal-feedback discrepancy. Finally, determination of goal discrepancy is likely to be made by prefrontal circuits. Fuster (1980) and Goldman–Rakic (1987) have studied activity in prefrontal neurons during the delay between stimulus and response. Their work suggests that these neurons are responsible for representations that guide complex responses. Similarly, developmental studies suggest that prefrontal cortex is involved in maintaining representations that inhibit reflexive or habitually reinforced behaviors to attain a goal (Diamond, 1990a). Cohen and Servan–Schreiber themselves note that dopamine may help augment contextual representations, which leads to more effective control over dominant response tendencies.

Furthermore, we would argue that there is phenomenological, neuropsychological, and biological evidence for using Cohen and Servan–Schreiber's network to model OCD. As discussed earlier, inadequate determination of goal discrepancy may be a central feature of OCD. Furthermore, it turns out

that neuropychological tests with the Stroop and the MFFT in OCD both reveal a subpopulation of patients with high error scores (Hollander, Schiffman et al., 1990). Finally, there is evidence that prefrontal and dopaminergic circuits are involved in OCD (Goodman et al., 1990; Khanna, 1988). It is notable that in animal and human models of prefrontal lobe damage, memory tasks that involve delay result in a bias toward past associative responses, which are task inappropriate and perseverative (Diamond, 1990b). Similarly, chronic administration of dopamine agonists may lead to stereotypies in animal models, and may lead to exacerbation of OCD in humans (Stein, Shoulberg, Helton, and Hollander, 1992).

What about the assessment of goal discrepancy as harmful in OCD? One place to start is to consider neural networks concerned with pattern switching. Pattern switching can be conceptualized in terms of behavioral inhibition/activation and harm avoidance. Thus a high threshold for pattern switching with implementation of only a single response pattern may be seen as equivalent to behavioral inhibition and high harm avoidance. In contrast, a low threshold for pattern switching with execution of several response patterns may be seen as equivalent to behavioral activation and low harm avoidance.

In our earlier discussion we noted that networks tend to converge toward states that maximize a global measure of constraint satisfaction. However, in order to ensure that a local maximum is in fact the global maximum, a network may include stochastic adjustments to individual units. This theoretical consideration is directly relevant to the question of how thresholds for pattern switching are set in the brain.

One possibility is that serotonin plays a role here. There is good evidence that serotonin is a central neuromodulator in determining behavioral inhibition/activation and harm avoidance (Soubrie, 1986; Stein, Hollander, and Liebowitz, 1993). Changes in serotonin activity may be associated with the modulation of thresholds for pattern switching. It is possible to speculate that serotonin is responsible for the random noise that allows neurons to shift away from maxima. Perhaps, when serotonin is low, there is increased noise, allowing for little adherence to any one particular response pattern, whereas when serotonin is high, there is decreased noise, allowing for little movement from any one particular response pattern. Whereas the prefrontal lobes are involved with goal selection, various subcortical structures, such as the limbic system, may mediate behavioral inhibition/activation or goal execution (Hestenes, 1992).

Indeed, Hestenes (1992) suggested that deficiencies in serotonin may lead to instabilities of pattern formation in psychotic illness. Instabilities in the selection of plans for speech output will produce frequent, sudden switches in meaning. Inability to decide among multiple interpretations of sensory stimuli will lead to perceptual confusion and hallucinations.

Similarly, a neural network that models a low threshold of pattern switching may be applied in conceptualizing OCD. Again this suggestion can be supported in terms of phenomenology, neuropsychology, and biology. First, we have already argued that exaggerated doubt/harm avoidance is a feature of OCD. Second, a behavioral inhibition pattern is seen on neuropsychological tests such as the MFFT, where a subgroup of OCD patients has a slow and correct response. Finally, there is good evidence that serotonin, which appears to have a central role in harm avoidance and behavioral inhibition, is crucial to the neurobiological mediation of OCD.

We suggest, then, that neural networks for the determination of goal discrepancy and for pattern switching allow a reformulation of many important features of OCD. Further work needs to be done to understand interaction of these networks, which are unlikely to be as clearly distinguishable as the previous discussion suggests. Indeed, there is biological evidence for interaction of neurotransmitter circuits in both normals and OCD. Thus serotonergic and dopaminergic neurons have significant interconnections (DeSimoni et al., 1987). We have found that response of plasma homovanillic acid, a metabolite of dopamine, to fenfluramine, a serotonin releaser and reuptake inhibitor, is decreased in OCD, but not in normals (Hollander, Stein et al., 1992). Better understanding of these interactions may lead to a rigorous neural network model of OCD.

Discussion

We have previously suggested that central psychological dysfunctions in OCD are determination and assessment of goal discrepancy, and that these dysfunctions have biological underpinnings. In this paper we discussed neural networks that can be employed to conceptualize these dysfunctions. We argued that the application of these networks to OCD is supported by phenomenological, neuropsychological, and biological data.

An important advantage of cognitive science is that it provides an integrative framework which incorporates different findings and methods from psychology and biology (Stein, 1992a). Similarly, a cognitive science model of OCD appears more comprehensive than purely psychoanalytic, biological, or learning models of OCD (Stein and Hollander, 1992). Connectionism provides cognitive science models with an additional degree of rigor insofar as it necessitates the inclusion of a "bottom–up" approach.

Connectionism has, however, been criticized by biologists for not incorporating neuroscience findings with sufficient exactness. In our discussion of models for OCD, although biological findings are addressed, there is a lack of detailed attention to neurobiology. For example, the model detailed by Cohen and Servan–Schreiber (1992) relies on back-propagation, a process which is

not seen in real neurons. Nevertheless, connectionist models also have value insofar as they address different levels of reality than do neurobiological models. To some extent, then, the biological plausibility of a particular network process is not necessarily paramount. As Cohen and Servan–Schreiber (1992) point out, the phenomenon of gradual descent — the gradual reduction of error by incremental adjustments in connection weights — is often seen in biological systems. Thus although back-propagation may not be a biological algorithm, it does describe biological learning.

Nevertheless, the connectionist approach described here does require further work on several fronts. Several important phenomena in OCD, such as the particular defenses that manifest in OCD patients, and the issues of control that are frequently seen in OCD, are not straightforwardly explicable in terms of deficits in goal discrepancy determination and assessment. The reason for the specificity of OCD symptoms, which may often have to do with grooming behaviors (Stein, Shoulberg et al., 1992) is not addressed. In addition, as discussed earlier, the division of OCD patients into subgroups with distinct biopsychological deficits is a preliminary research strategy. Finally, and perhaps most importantly, more detailed work on the computational implementation and integration of the networks described here is necessary.

We suggest that despite these drawbacks, our connectionist model of OCD tackles several key features of the disorder, and brings together several areas that have previously been difficult to integrate. Furthermore, a connectionist model of OCD provides a heuristic for further theoretical and empirical research on this complex and poorly understood disorder.

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